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What Do We Really Know About "tinea incognita"?

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SUMMARY The term "tinea incognita" refers to diverse clinical presentation of mycotic infections modified by inappropriate use of topical or systemic corticosteroids. A 67-year-old male patient with a five-year history of generalized erythematous plaques on the trunk and extremities, previously treated with topical corticosteroids, is described. The lesions mainly showed a psoriasiform, some eczematous appearance, few of them showing a clinical picture of folliculitis. The native mycologic specimen was negative. The diagnosis was made on the basis of mycologic culture finding of *Trichophyton interdigitale* growth. Systemic and topical antimycotic therapy administered for two months resulted in complete regression of skin lesions.

KEY WORDS: Tinea incognita; *Trichophyton mentagrophytes*; *Trichophyton interdigitale*; chronic infection

INTRODUCTION

The term "tinea incognita" refers to dermatophytic skin infections with a relatively characteristic clinical picture of annular or oval erythematous lesions sometimes large in diameter, with marked scaly margins, modified by use of topical or systemic corticosteroids (1). Tinea incognita may mimic the clinical picture of various dermatoses, thus posing an additional differential diagnostic problem. The diagnosis is based both on history and clinical picture, and generally confirmed by laboratory mycologic testing including native microscopy and culture. Pathohistologic examination is not routinely performed because the aforementioned procedures usually prove adequate for diagnosis verification (2). However, pathohistology is very useful in less clear cases. Mycelium elements may be visualized by various staining techniques such as periodic-acid-Schiff (PAS) or silver impregnation according to Grocott (2). The management of tinea incognita generally includes systemic antimycotic treatment (3). The course of the disease may be chronic due to modification of the typical clinical picture of dermatophytosis by topical corticosteroid therapy. The prognosis of the disease generally is highly favorable, as antimycotic therapy nearly always results in complete recovery (4).

CASE REPORT

Some 20 years before, a male patient now aged 67 observed desquamation and erythema involving the interdigital spaces of his feet. Five years before presentation, dissemination of the lesions in part showing eczematous and psoriasiform appearance, some showing a clinical picture of folliculitis, occurred. The lesions involved almost the entire surface of the abdomen (Fig. 1) and lower extremities (Fig. 2). At the very onset of skin lesions, the patient applied topical cortico-

steroids (betamethasone + salicylic acid cream), which resulted in partial regression of the lesions, i.e. reduction of inflammatory erythema and scaling. Exacerbations of the disease occurred over the five consecutive years, the clinical picture being most pronounced in summer. Otherwise, the skin surface was free from any major alterations except for generalized toe nail discoloration and dystrophy, red and macerated, eroded and inflamed interdigital spaces, and minor dandruff on the scalp. Mycologic finding was negative on three occasions, whereas culture showed the presence of *Trichophyton interdigitale*. Skin biopsy revealed nonspecific inflammatory changes, folliculitis and perifolliculitis (Figs. 3, 4 and 5).

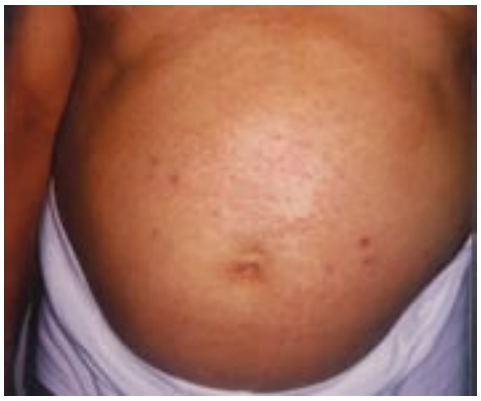

Figure 1. Clinical picture: eczematous, psoriasiform and folliculitis-like skin lesions in the abdominal region.

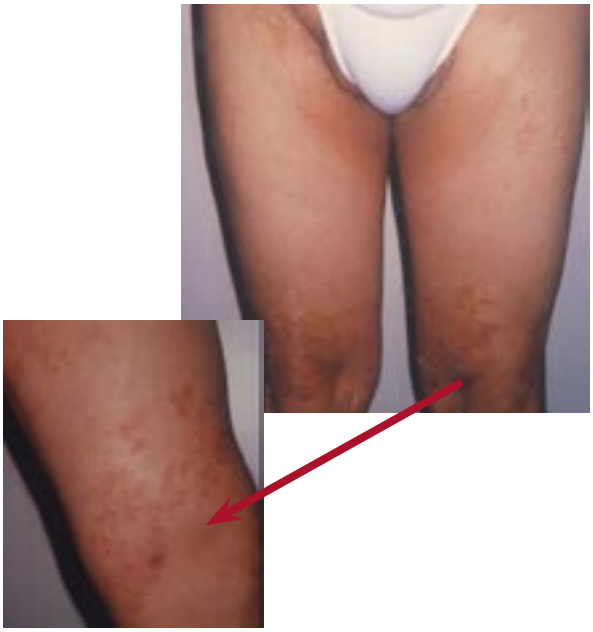

Figure 2. Clinical picture: the same picture is seen in lower extremities.

Blood and urine biochemistry test results were normal; blood glucose 5.3 mmol/l; peripheral blood immunophenotyping yielded normal finding: CD3+ (T) 77%; CD3+CD4+ 47%; CD3+CD8+ 31%; CD4/CD8 1.52%; CD3-CD 16+56 (NK) 14%; CD3-CD19+ (B) 11%; functional lymphocyte tests

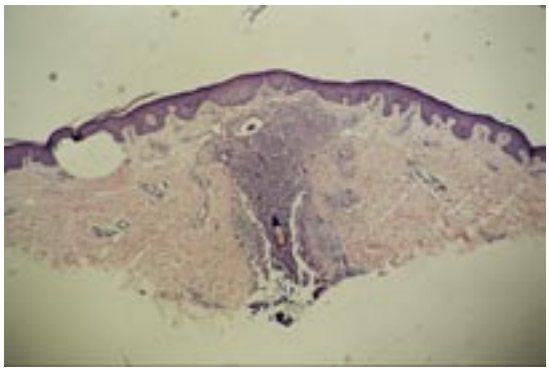

Figure 3. Perifolliculitis – a mixed perifollicular infiltrate of mononuclear cells and neutrophils (hematoxylin-eosin stain; original magnification x25).

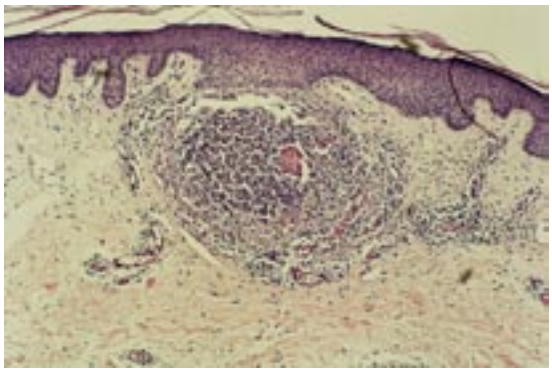

Figure 4. Perifolliculitis – detail (hematoxylin-eosin stain; original magnification x50).

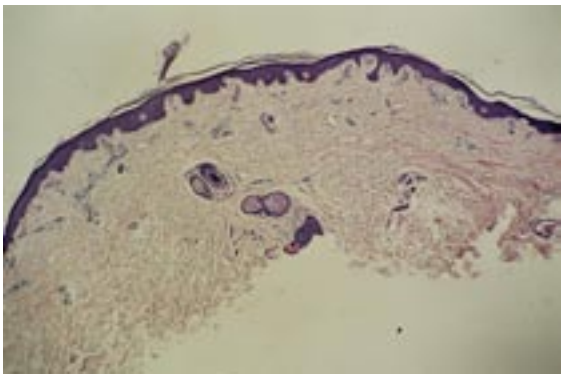

Figure 5. Scanty subepidermal perivascular infiltrate of mononuclear cells (hematoxylin-eosin stain; original magnification x25).

Figure 6. Two-month therapy resulted in complete regression of abdominal skin lesions.

Figure 7. The lesions on lower extremities also showed complete regression, with residual scar after skin biopsy.

(mitogenic) were normal: PHA 18.1, Con A 15.1, PWM 12.6, PPd 193; analysis of respiratory burst in granulocytes by flow cytometry also produced normal finding. IgG anti-HIV1 and IgG anti-HIV2 findings were negative. A systemic antimycotic treatment with terbinafine, 250 mg daily, and miconazole cream topically was initiated. Two-month therapy resulted in complete regression of the skin lesions (Figs. 6 and 7). Hepatogram and the levels of cholesterol and triglycerides were normal both before and after terbinafine therapy.

DISCUSSION

The presented case is an example of an immunocompetent individual in whom a generalized, unrecognized dermatomycosis was for five years treated as psoriasis with topical corticosteroids. Corticosteroids are potent agents, and their effects in the management of skin diseases include decreased epidermal mitotic activity, epidermal thinning, decrease in epidermal cell size, decreased overall intraepidermal metabolic activity, dermal thinning and decreased collagen synthesis (4), and reduction of inflammation (4,5). As they reduce inflammatory reaction, corticosteroids are widely used for radical treatment of many inflammatory dermatoses (4). Changes in the clinical picture are especially pronounced in case of inappropriate use of corticosteroids in the management of unrecognized dermatophytoses (4). Proper understanding of dermatophytic infections requires, among others, thorough knowledge of their ecology (6). Some etiologic species predominantly reside in the soil (geophilic), on animals (zoophilic), or on humans (anthropophilic) (6,7). The geophilic and zoophilic species may sporadically infect humans. The zoophilic species cause a more inflammatory response, sometimes with a suppurative clinical picture, primarily on the protected and unprotected skin areas having been in direct contact with the soil and animal, respectively (3,6). The anthropophilic species cause inflammatory reaction of lower severity, mostly localized on the protected skin areas (groins, knee, foot), and are usually transmitted by direct contact between the infected and healthy persons or indirectly through fomites (6). The severity of the clinical picture greatly depends on the host's health status, primarily on concomitant diseases such as diabetes mellitus, malignant diseases of the lymphatic system, immunodeficiency, HIV/AIDS, Cushing's syndrome, etc. (6). The host's age, sex, race, lifestyle, occupation and geographical location also play an important role (6). Additional factors that stimulate the development of dermatophytosis include mechanical trauma compromising the protective role of the skin, hyperhidrosis, occlusive effect of the clothes, etc. (6). During the period of incubation, dermatophytes grow in the horny layer metabolizing keratin they feed on (6). Their presence in the horny layer evokes eczematous response in the underlying epidermis (5). Eczematous skin is a poor keratin producer, thus leaving the fungi without food supply and resulting in spontaneous disappearance of the infection (5). At the same time, the use of topical corticosteroids stimulates fungal growth by suppressing the local immune response, thus allowing for the occurrence of more virulent strains (5). Accordingly, corticosteroids reduce resistance to infection, in this case mediated by cellular immunity. This in turn increases the likelihood for the infection to proceed undiagnosed, additionally increasing the patient's susceptibility to infection. The period of incubation is usually free from

any clinical signs of infection, and dermatophytes can only be demonstrated in a native specimen (6). A typical clinical picture with annular erythematous foci, scaling and pustules on the periphery, and intensive pruritus develops between day 10 and 35 in persons with dermatophyte infection for the first time, whereas reinfection in the same person produces a very early inflammatory response (6). Clinically, tinea incognita may mimic various dermatoses such as erythema chronicum migrans (8), contact dermatitis (3), psoriasis (3), lupus erythematosus (3,9), polymorphous light eruption (10), which thus frequently remain unrecognized and mistreated. Dermatophytosis on the face may also frequently be misdiagnosed, mimicking the clinical picture of seborrheic dermatitis (11,12), contact dermatitis (11,12), lupus erythematosus (11,12), photosensitive dermatitis (11,12) and rosacea (12), especially in adults, with papules, pustules and patches of erythema, with or without scaling, and with absence of typical annular lesions (12). Patients with this problem consistently report on previous treatment of the disease with topical or systemic corticosteroids (4), prescribed by the physician or, by no means infrequently, as self-treatment, as some corticosteroids are also available as over-the-counter (OTC) agents (11). It should be borne in mind that a dermatophytic infection may be overlooked in psoriatics using topical corticosteroids (4), which especially applies to patients with inverse psoriasis in whom newly developed erythema and scaling lesions may be considered and treated as psoriasis with topical corticosteroids, while being actually underlain by ringworm infection rather than psoriasis. Therefore, making the diagnosis exclusively on the basis of clinical picture may result in inappropriate topical corticosteroid therapy, which visibly reduces infiltration and scaling in dermatophytoses but the symptoms will recur soon after therapy discontinuation. In chronic dermatophytosis, the reservoir of infection are dermatophytes found in hair follicles (9), reaching them during the process of hair keratin fermentation. In our patient, the histologic picture was predominated by folliculitis, whereas specific staining failed to demonstrate fungal elements either in the follicles or in the horny layer. We are inclined to believe that follicles served as a reservoir of infection, disseminating to the groin and abdominal region by autoinoculation from interdigital spaces of the feet. That is why some authors suggest that stockings be put on first, before all other clothes (7). In our patient, the chronic course of the disease was, among others, induced by the lesions having been caused by an anthropophilic species (*T. interdigitale*), which induces weak inflammatory response that was additionally suppressed by longterm corticosteroid therapy. The exact mechanisms by which some immunocompetent persons like our patient develop persistent and recurrent dermatophytic skin infection have not yet been fully clarified. Some experimental studies have shown that persistent infection occurs due to the secondary local inhibitory effect of dermatophytic antigens *in vivo* or to selective anergy to dermatophyte antigens (13). The role of specific mechanisms of defense is not clear, however, cellular immunity is known to play a more important role than humoral immunity (2), the latter still awaiting full elucidation. Patients with extensively disseminated dermatophytosis have high titers of IgG, IgM and IgA antibodies, which are not dermatophyte specific (6). A high IgE titer indicates a chronic course of the disease and is frequently detected in associated atopy (14). It has been speculated that the occurrence of infection could be explained by local or systemic modulation of T lymphocyte activity as well as by activation of helper lymphocytes (14). In our patient, both basic and specific diagnostic tests failed to confirm the disease, i.e. immunosuppression supporting dermatophytic infection. Positive culture is the gold standard for accurate diagnosis, however, the diagnosis is verified by the finding of hyphae or spores in native specimen, or mycelial elements in pathohistologic specimen (2). The treatment with systemic antimycotics has to be initiated when the mycotic infection is proved (5).

CONCLUSION

Mycotic infection should always be considered in case of persistent and recurrent skin lesions resembling other dermatoses. Thorough history, careful laboratory testing with particular reference to mycologic culture findings, and skin biopsy in vague cases are necessary procedures to reach an accurate diagnosis of dermatophytic skin infection. Some experienced dermatologists suggest that mycologic examination should only be done in two instances, i.e. when the clinical picture is typical for fungal skin infection, and when it bears no such resemblance at all.

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